

National Library of Medicine - Medical Subject Headings

2005 MeSH

MeSH Supplementary Concept Data

[Return to Entry Page](#)

Name of Substance	fluoresceinyl-arginyl-glutamyl-aspartyl-glutamyl-aspartyl-glutamyl-isoleucyl-glutamyl-tryptophan
Record Type	C
Registry Number	0
Entry Term	fluoresceinyl-REDEDEIEW
Entry Term	fluoresceinyl-Arg-Glu-Asp-Glu-Asp-Glu-Ile-Glu-Trp
Entry Term	Fl-CDB3
Heading Mapped to	* Fluoresceins
Heading Mapped to	* Oligopeptides
Source	Proc Natl Acad Sci U S A 2003 Nov 11;100(23):13303-7
Frequency	1
Note	binds and stabilizes the tumor suppressor p53 core domain, thereby preventing denaturation; may serve as an antineoplastic agent
Date of Entry	20031226
Unique ID	C479932

[Return to Entry Page](#)

[Link to NLM Cataloging Classification](#)

Refine Search

Search Results -

Term	Documents
@PY	8218647
(20 AND (@PY < "2001")).PGPB,USPT.	22
(L20 AND @PY<2001).PGPB,USPT.	22

Database:

US Pre-Grant Publication Full-Text Database
 US Patents Full-Text Database
 US OCR Full-Text Database
 EPO Abstracts Database
 JPO Abstracts Database
 Derwent World Patents Index
 IBM Technical Disclosure Bulletins

Search:

L21

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Search History

DATE: Tuesday, March 08, 2005 [Printable Copy](#) [Create Case](#)

Set Name Query
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Hit Count Set Name
 result set

DB=PGPB,USPT; THES=ASSIGNEE; PLUR=YES; OP=ADJ

<u>L21</u>	L20 and @py<2001	22	<u>L21</u>
<u>L20</u>	L19 and L5	200	<u>L20</u>
<u>L19</u>	binding protein same p53	851	<u>L19</u>
<u>L18</u>	L17 and p53	77	<u>L18</u>
<u>L17</u>	L16 and @py<2001	77	<u>L17</u>
<u>L16</u>	L15 and stabil\$	77	<u>L16</u>
<u>L15</u>	L14 and denaturation	122	<u>L15</u>
<u>L14</u>	L13 and tumor	412	<u>L14</u>
<u>L13</u>	L12 and @py<2001	480	<u>L13</u>
<u>L12</u>	L5 and L8	3625	<u>L12</u>
<u>L11</u>	L10 and L9 and L8 and L5	0	<u>L11</u>
<u>L10</u>	prevent denaturation	430	<u>L10</u>

<u>L9</u>	antineoplastic agent	4423	<u>L9</u>
<u>L8</u>	p53	11124	<u>L8</u>
<u>L7</u>	L6 and tumor sepressor	0	<u>L7</u>
<u>L6</u>	L5 and p53	3625	<u>L6</u>
<u>L5</u>	Fluoresceins	33488	<u>L5</u>
<u>L4</u>	fluoresceinyl-Arg-Glu-Asp-Glu-Asp-Glu-Ile-Glu-Trp	0	<u>L4</u>
<u>L3</u>	Fluoresceinyl-REDEDEIEW	0	<u>L3</u>
<u>L2</u>	REDEDEIEW	1	<u>L2</u>
<u>L1</u>	CDB3	7	<u>L1</u>

END OF SEARCH HISTORY

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NEWS	12	DEC 17	CERAB reloaded; updating to resume; current-awareness alerts (SDIs) affected
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NEWS	15	DEC 30	CAPLUS - PATENT COVERAGE EXPANDED
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NEWS	17	FEB 25	CA/CAPLUS - Russian Agency for Patents and Trademarks (ROSPATENT) added to list of core patent offices covered
NEWS	18	FEB 10	STN Patent Forums to be held in March 2005
NEWS	19	FEB 16	STN User Update to be held in conjunction with the 229th ACS National Meeting on March 13, 2005
NEWS	20	FEB 28	PATDPAFULL - New display fields provide for legal status data from INPADO
NEWS	21	FEB 28	BABS - Current-awareness alerts (SDIs) available
NEWS	22	FEB 28	MEDLINE/LMEDLINE reloaded
NEWS	23	MAR 02	GBFULL: New full-text patent database on STN
NEWS	24	MAR 03	REGISTRY/ZREGISTRY - Sequence annotations enhanced
NEWS	25	MAR 03	MEDLINE file segment of TOXCENTER reloaded
NEWS EXPRESS			JANUARY 10 CURRENT WINDOWS VERSION IS V7.01a, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 10 JANUARY 2005
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FILE COVERS 1907 - 8 Mar 2005 VOL 142 ISS 11
FILE LAST UPDATED: 7 Mar 2005 (20050307/ED)

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> s friedler a/au
L1 3 FRIEDLER A/AU

=> d L1 1-3 ibib,abs

L1 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2002:101989 CAPLUS

DOCUMENT NUMBER: 136:303881

TITLE: Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin

AUTHOR(S): Knowler, William C.; Barrett-Connor, Elizabeth; Fowler, Sarah E.; Hamman, Richard F.; Lachin, John M.; Walker, Elizabeth A.; Nathan, David M.; Bray, G. A.; Culbert, I. W.; Champagne, C. M.; Crow, M. D.; Dawson, L.; Eberhardt, B.; Greenway, F. L.; Guillory, F. G.; Herbert, A. A.; Jeffirs, M. L.; Joyce, K.; Kennedy, B. M.; Lovejoy, J. C.; Mancuso, S.; Melancon, L. E.; Morris, L. H.; Reed, L.; Perault, J.; Rau, K.; Ryan, D. H.; Sanford, D. A.; Smith, K. G.; Smith, L. L.; Smith, S. R.; St. Amant, J. A.; Terry, M.; Tucker, E.; Tulley, R. T.; Vicknair, P. C.; Williamson, D.; Zachwieja, J. J.; Ehrmann, D. A.; Matulik, M. J.; Clark, B.; Collins, D. A.; Czech, K. B.; DeSandre, C.; Geiger, G.; Frief, S.; Harding-Clay, B.; Hilbrich, R. M.; Le Grange, D.; McCormick, M. R.; McNabb, W. L.; Polonsky, K. S.; Sauter, N. P.; Semenske, A. R.;

Stepp, K. A.; Tobian, J. A.; Watson, P. G.; Mendoza, J. T.; Smith, K. A.; Caro, J.; Goldstein, B.; Lark, C.; Menefee, L.; Murphy, L.; Pepe, C.; Spandorfer, J. M.; Goldberg, R. B.; Rowe, P.; Calles, J.; Casanova, P.; Donahue, R. P.; Florez, H. J.; Giannella, A.; Larreal, G.; McLymont, V.; Mendez, J.; O'Hara, P.; Ojito, J.; Prineas, R.; Saab, P. G.; Haffner, S. M.; Montez, M. G.; Lorenzo, C.; Miettinen, H.; Mobley, C. M.; Mykkanen, L. A.; Rozek, M. M.; Hamman, R. F.; Nash, P. V.; Testaverde, L.; Anderson, D. R.; Ballonoff, L. B.; Bouffard, A.; Calonge, B. N.; Farago, M.; Georgitis, W. J.; Hill, J. O.; Hoyer, S. R.; Jortberg, B. T.; Merenich, J. A.; Miller, M.; Regensteiner, J. G.; Seagle, H. M.; Smith, C. M.; Steinke, S. C.; Van Dorsten, B.; Horton, E. S.; Lawton, K. E.; Arky, R. A.; Bryant, M.; Burke, J. P.; Caballero, E.; Callaghan, K. M.; Devlin, D.; Franklin, T.; Ganda, O. P.; **Goebel-Fabbri, A. E.**; Harris, M.; Jackson, S. D.; Jacobsen, A. M.; Kula, L. M.; Kocal, M.; Ledbury, S.; Malloy, M. A.; Mullooly, C.; Nicosia, M.; Oldmixon, C. F.; Pan, J.; Pomposelli, C.; Quitongan, M.; Rubtchinsky, S.; Schweizer, D.; Seely, E. W.; Simonson, D.; Smith, F.; Solomon, C. G.; Tyson, J.; Warram, J.; Kahn, S. E.; Montgomery, B. K.; Alger, M.; Allen, E.; Barrett, T.; Bhanji, D.; Cowan, J.; Cullen, J.; Fujimoto, W. Y.; Katz, B.; Knopp, R. H.; Lipkin, E. W.; Marr, M.; McCann, B. S.; Palmer, J. P.; Schwartz, R. S.; Uyema, D.; Kitabachi, A. E.; Murphy, M. E.; Applegate, W. B.; Bryer-Ash, M.; Coble, J. H.; Crisler, A.; Cunningham, G.; Franklin, A. W.; Frieson, S. L.; Green, D. L.; Imseis, R.; Kennedy, C. L.; Lambeth, H. C.; Latif, K. A.; Lichtermann, L. C.; McIntyre, M. D.; Nault, J. D.; Oktaei, H.; O'Toole, M. L.; Ricks, H.; Rutledge, L. M. K.; Schussler, S. C.; Sherman, A. R.; Smith, C. M.; Soberman, J. E.; Stewart, K. J.; Van Brunt, D. L.; Williams-Cleaves, B. J.; Johnson, M. K.; Behrends, C.; Cook, M. L.; Fitzgibbon, M.; Giles, M. M.; Heard, D.; Johnson, C.; Larsen, D.; Lowe, A.; Lyman, M.; McPherson, D.; Molitch, M. E.; Pitts, T.; Reinhart, R.; Roston, S.; Schinleber, P. A.; Nathan, D. M.; McKittrick, C.; Abbott, K.; Anderson, E.; Bissett, L.; Cagliari, E.; Crowell, S.; Delahanty, L.; Fritz, S.; Hayward, K.; Levina, E.; Michel, T.; Norman, D.; O'Keefe, J.; Poulos, A.; Ronan, L.; Rosal, M.; Salerno, M.; Schneider, M.; Shagensky, C.; Steiner, B.; Turgeon, H.; Young, A.; Olefsky, J. M.; Carrion-Petersen, M. L.; Barrett-Connor, E.; Beltran, M.; Caenepeel-Mills, K.; Edelman, S. V.; Ford, R. O.; Garcia, J.; Hagerty, M.; Henry, R. R.; Hill, M.; Horne, J.; Leos, D.; Matney, J.; Mudaliar, S.; Petersen, G.; Pollard, A.; Polonsky, W.; Szerdi, S.; Torio-Hurley, J.; Vejvoda, K.; Pi-Sunyer, F. X.; Lee, J. E.; Allison, D. B.; Agharanya, N.; Aronoff, N. J.; Baldo, M.; Foo, S. T.; Hagamen, S.; Pal, C.; Parkes, K.; Pena, M.; Van Wye, G. E. H.; Marrero, D. G.; Kukman-Kelly, M. S.; Dorson, Y. F.; Fineberg, S. E.; Guare, J. C.; Hadden, A.; Hills, B.; Ignaut, J. M.; Jackson, M. A.; Kirkman, M. S.; Mather, K.; McAree, G.; Porter, B. D.; Prince, M. J.; Wheeler, M. L.; Ratner, R. E.; Youssef, G.; Shapiro, S.; Bonar, A.; Bronsord, M.; Brown, E.; Cheatham, W. W.; Cola, S.; Comfort, A.; Boggs, G.; Eagle, C.; Evans, C.; Gorman, E.; Johnson, R.; Levetan, C.; Kellum, T.; Lagarda, M.; Nair, A. K.;

Passaro, M. D.; Phillips, W.; Saad, M. F.; Budgett, M.; Fahmi, S.; Jinagouda, S. D.; Bernaba, B.; Bodkin, S. L.; Ciobanu, V.; Commisso, R.; Cosenza, C.; Dinh, T.; Gonzalez, M.; Khan, A.; Kumar, D.; Lui, G.; Mehra, V.; Sharma, A.; Soukiazian, S.; Szamos, K.; Tramanian, A.; Vargas, A.; Zambrana, N.; White, N. H.; Santiago, A. S.; Das, S.; Brown, A. L.; Dagogo-Jack, S.; Fisher, E. B.; Hurt, E.; Jones, T.; Kerr, M.; Ryder, L.; Santiago, J. V.; Wernimont, C.; Saudek, C. D.; Bradley, V. L.; Fowlkes, T.; Joseph, H.; Brancati, F. L.; Charleston, J. B.; Clark, J. M.; Horak, K.; Jiggetts, D.; Mosley, H.; Rubin, R. R.; Samuels, A.; Stewart, K. J.; Thomas, L.; Williamson, P.; Schade, D. S.; Adams, K. S.; Adler, L. F.; Bland, A.; Bowling, D. A.; Boyle, P. J.; Burge, M. R.; Butler, L.; Canady, J. L.; Chai, L.; Colleran, K. M.; Guillen, M.; Gonzales, Y.; Gutierrez, M.; Hornbeck, D.; Johannes, C.; Karz, P.; King, C.; Libby, E. N., III; McCalman, R.; Montoya, D. A.; Rassam, A.; Rubinchik, S.; Senter, W.; Shamoan, H.; Brown, J. O.; Adames, J.; Blanco, E.; Cox, L.; Crandall, J. P.; Duffy, H.; Engel, S.; Friedler, A.; Harroun, T.; Howard-Century, C. J.; Kloiber, S.; Longchamp, N.; Pompei, D.; Violino, E.; Walker, E. A.; Wylie-Rosett, J.; Zimmerman, E.; Zonszein, J.; Wing, R. R.; Kramer, M. K.; Barr, S.; Boraz, M. A.; Clifford, L.; Culyba, R.; Frazier, M.; Gilligan, R.; Harris, L.; Harrier, S.; Henderson, W.; Jeffreis, S.; Koenning, G.; Kriska, A. M.; Maholic, K.; Manjoo, Q.; Mullen, M.; Noel, A.; Orchard, T. J.; Orro, A.; Semler, L. N.; Smith, C.; Smith, M.; Stapinski, V.; Viteri, J.; Wilson, T.; Williams, K. V.; Zgibor, J.; Arakaki, R. F.; Latimer, R. W.; Baker-Ladao, N. K.; Beddow, R. M.; Braginsky, R.; Calizar, M.; Dias, L. M.; Durham, N.; Dupont, D. A.; Fukuhara, L. L.; Inouye, J.; Mau, M. K.; Mikami, K.; Mohideen, P.; Odom, S. K.; Sinkuie-Kam, B.; Tokunaga, J. S.; Twiggs, R. U.; Wang, C. Y.; Vita, J.; Knowler, W. C.; Coeeyate, N. J.; Hoskin, M. A.; Percy, C. A.; Acton, K. J.; Andre, V. L.; Antone, S.; Baptisto, N. M.; Barber, R.; Segay, S.; Bennett, P. H.; Benson, M. B.; Beyale, S.; Bird, E. C.; Broussard, B. A.; Chavez, M.; Daeawyma, T. S.; Doughty, M. S.; Duncan, R.; Edgerton, C.; Ghahate, J. M.; Glass, M.; Gohdes, D.; Grant, W.; Hanson, R. L.; Horse, E.; Hughte, G.; Ingraham, L. E.; Jackson, M. C.; Jay, P. A.; Kaskalla, R. S.; Kessler, D.; Kobus, K. M.; Krakoff, J.; Manus, C.; Morgan, T.; Nashboo, Y.; Nelson, J.; Pauk, G. L.; Poirier, S.; Polczynski, E.; Reidy, M.; Roumain, J.; Rowse, D. H.; Roy, R. J.; Sangster, S.; Sewemaenewa, J.; Tonemah, D.; Wilson, C.; Yazzie, M.; Fowler, S.; Brenneman, T.; Abebe, S.; Bain, R.; Bamdad, J.; Callaghan, J.; Edelstein, S. L.; Gao, Y.; Grimes, K. L.; Grover, N.; Hirst, K.; Jones, S.; Jones, T. L.; Katz, R. J.; Lachin, J. M.; Orlosky, R.; Stimpson, C. E.; Suiter, C.; Temprosa, M. G.; Walker-Murray, F. E. M.; Garfield, S.; Eastman, R.; Fradkin, J.; Andres, R.; Engelgau, M. M.; Venkat Narayan, K. M.; Williamson, D. F.; Herman, W. H.; Marcovina, S. M.; Aldrich, A.; Chandler, W. L.; Rautaharju, P. M.; Pemberton, N. T.; Prineas, R.; Rautaharju, F. S. R.; Zhang, Z.; Mayer-Davis, E. J.; Costacou, T.; Martin, M.; Sparks, K. L.; O'Leary, D. H.; Funk, L. R. C.; O'Leary, K. A.; Polak, J. F.; Stamm, E. R.; Scherzinger, A. L.; Wing, R. R.; Gillis, B. P.;

Huffmyer, C.; Kriska, A. M.; Venditti, E. M.; Walker, E. A.; Harroun, T.; Ganiats, T. G.; Groessl, E. J.; Beerman, P. R.; David, K. M.; Kaplan, R. M.; Sieber, W. J.; Genuth, S. M.; Cahill, G. F.; Ferris, F. L., III; Gavin, J. R., III; Halter, J. B.; Wittes, J.; Henry, R. R.; Haffner, S. M.; Rubin, R. R.; Montgomery, B. K.; Ratner, R. E.; Herman, W. H.; Kahn, S. E.; Santiago, J. V.; Olefsky, J.; Wing, R. R.; Saudek, C.; Montez, M.; Kramer, K.; Hamman, R. F.; Knowler, W. C.; Goldberg, R. B.; Fujimoto, W. Y.; Charleston, J.; Nathan, D. M.

CORPORATE SOURCE:

Diabetes Prevention Program Coordinating Center,
Washington Univ., Rockville, MD, 20852, USA

SOURCE:

New England Journal of Medicine (2002), 346(6),
393-403

CODEN: NEJMAG; ISSN: 0028-4793

PUBLISHER:

Massachusetts Medical Society

DOCUMENT TYPE:

Journal

LANGUAGE:

English

AB Type 2 diabetes affects approx. 8 % of adults in the United States. Some risk factors - elevated plasma glucose concns. in the fasting state and after an oral glucose load, over-weight, and a sedentary lifestyle - are potentially reversible. We hypothesized that modifying these factors with a lifestyle-intervention program or the administration of metformin would prevent or delay the development of diabetes. We randomly assigned 3234 nondiabetic persons with elevated fasting and post-load plasma glucose concns. to placebo, metformin (850 mg twice daily), or a lifestyle-modification program with the goals of at least a 7 % weight loss and at least 150 min of phys. activity per wk. The mean age of the participants was 51 yr, and the mean body-mass index (the weight in kilograms divided by the square of the height in meters) was 34.0; 68 % were women, and 45 % were members of minority groups. The average follow-up was 2.8 yr. The incidence of diabetes was 11.0, 7.8, and 4.8 cases per 100 person-years in the placebo, metformin, and life-style groups, resp. The lifestyle intervention reduced the incidence by 58 % (95 % confidence interval, 48 to 66 %) and metformin by 31 % (95 % confidence interval, 17 to 43 %), as compared with placebo; the lifestyle intervention was significantly more effective than metformin. To prevent one case of diabetes during a period of three years, 6.9 persons would have to participate in the lifestyle-intervention program, and 13.9 would have to receive metformin. Lifestyle changes and treatment with metformin both reduced the incidence of diabetes in persons at high risk. The lifestyle intervention was more effective than metformin.

REFERENCE COUNT:

29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1999:578782 CAPLUS

DOCUMENT NUMBER: 132:11558

TITLE: Human immunodeficiency virus type 1 Vif-derived
peptides inhibit the viral protease and arrest virus
production

AUTHOR(S): Gilon, C.; Friedler, A.; Baraz, L.;
Blumenzweig, I.; Nussinov, O.; Steinitz, M.; Kotler,
M.

CORPORATE SOURCE: Department of Organic Chemistry, The Hebrew
University, Jerusalem, 91904, Israel

SOURCE: Peptide Science: Present and Future, Proceedings of
the International Peptide Symposium, 1st, Kyoto, Nov.
30-Dec. 5, 1997 (1999), Meeting Date 1997, 439-441.
Editor(s): Shimonishi, Yasutsugu. Kluwer: Dordrecht,
Neth.

CODEN: 68BYA5

DOCUMENT TYPE:

Conference

LANGUAGE: English

AB To study the effects of Vif derived peptides on HIV maturation and the autoprocessing of the Gag and Gag-Pol polyproteins, N terminal Vif polypeptides were synthesized and inhibition of viral proteases was assayed by ELISA. The results show that 4 out of the 11 peptides significantly inhibited viral proteases. Vif derived polypeptides also inhibited the autoprocessing of the Gag and Gag-Pol polyproteins in bacteria and eukaryotic cells. These data suggest that the Vif derived peptides form an attractive potential therapeutic agent for inhibition of HIV proteases during HIV-1 infection in humans.

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1999:578657 CAPLUS

DOCUMENT NUMBER: 132:102441

TITLE: BCvir: backbone cyclic peptide, which mimics the nuclear localization signal of human immunodeficiency virus type 1 matrix protein, inhibits nuclear import and virus production in non-dividing cells

AUTHOR(S): Friedler, A.; Zakai, N.; Karni, O.; Broder, Y. C.; Baraz, L.; Kotler, M.; Loyter, A.; Gilon, C.

CORPORATE SOURCE: Department of Organic Chemistry, Institute of Chemistry, The Hebrew University of Jerusalem, Jerusalem, 91904, Israel

SOURCE: Peptide Science: Present and Future, Proceedings of the International Peptide Symposium, 1st, Kyoto, Nov. 30-Dec. 5, 1997 (1999), Meeting Date 1997, 70-72. Editor(s): Shimonishi, Yasutsugu. Kluwer: Dordrecht, Neth.

CODEN: 68BYA5

DOCUMENT TYPE: Conference

LANGUAGE: English

AB A backbone cyclic NLS-mimetic peptide was found which inhibits nuclear import in invitro assay systems as well as HIV-1 replication in infected cultured cells. This peptide, BCvir, is resistant to proteolysis. BCvir and similar peptides are potential candidates for the development of a novel class of anti-viral drugs based on blocking nuclear import of viral genomes.

REFERENCE COUNT: 10 THERE ARE 10 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> s Friedler Assaf

5 FRIEDLER

22 ASSAF

L2 0 FRIEDLER ASSAF

(FRIEDLER(W)ASSAF)

=> s fluoresceinyl and pepetide and p53

119 FLUORESCINEYL

14 PEPETIDE

6 PEPETIDES

20 PEPETIDE

(PEPETIDE OR PEPETIDES)

30076 P53

L3 0 FLUORESCINEYL AND PEPETIDE AND P53

=> s fluoresceins

L4 227 FLUORESCINS

=> s binding peptide

857885 BINDING

1890 BINDINGS

```

      858414 BINDING
          (BINDING OR BINDINGS)
      325639 PEPTIDE
      238122 PEPTIDES
      416868 PEPTIDE
          (PEPTIDE OR PEPTIDES)
L5      3659 BINDING PEPTIDE
          (BINDING(W) PEPTIDE)

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=> s p53 and binding protein
      30076 P53
      857885 BINDING
          1890 BINDINGS
      858414 BINDING
          (BINDING OR BINDINGS)
      1719330 PROTEIN
      1192773 PROTEINS
      1996128 PROTEIN
          (PROTEIN OR PROTEINS)
      134380 BINDING PROTEIN
          (BINDING(W) PROTEIN)
L6      1376 P53 AND BINDING PROTEIN

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=> d his

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      FILE 'CAPLUS' ENTERED AT 08:51:08 ON 08 MAR 2005

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L1      3 S FRIEDLER A/AU
L2      0 S FRIEDLER ASSAF
L3      0 S FLUORESCEINYL AND PEPETIDE AND P53
L4      227 S FLUORESCEINS
L5      3659 S BINDING PEPTIDE
L6      1376 S P53 AND BINDING PROTEIN

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=> s L6 and L5 and L4
L7      0 L6 AND L5 AND L4

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=> s L4 and L6
L8      0 L4 AND L6

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=> s L6 and prevent denaturation
      221001 PREVENT
      93176 PREVENTS
      306930 PREVENT
          (PREVENT OR PREVENTS)
      34688 DENATURATION
          158 DENATURATIONS
      34731 DENATURATION
          (DENATURATION OR DENATURATIONS)
          72 PREVENT DENATURATION
          (PREVENT(W) DENATURATION)
L9      0 L6 AND PREVENT DENATURATION

```

```

=> L6 and stabilizing molecule
L6 IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

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=> s L6 and stabilising molecule
      80 STABILISING
      46847 MOLECULE
      145652 MOLECULES

```

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186045 MOLECULE
      (MOLECULE OR MOLECULES)
2198618 MOL
590871 MOLS
2520191 MOL
      (MOL OR MOLS)
2559007 MOLECULE
      (MOLECULE OR MOL)
0 STABILISING MOLECULE
      (STABILISING (W) MOLECULE)
L10      0 L6 AND STABILISING MOLECULE

```

=> d his

(FILE 'HOME' ENTERED AT 08:50:59 ON 08 MAR 2005)

FILE 'CAPLUS' ENTERED AT 08:51:08 ON 08 MAR 2005

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L1      3 S FRIEDLER A/AU
L2      0 S FRIEDLER ASSAF
L3      0 S FLUORESCCEINYL AND PEPETIDE AND P53
L4      227 S FLUORESCCEINS
L5      3659 S BINDING PEPTIDE
L6      1376 S P53 AND BINDING PROTEIN
L7      0 S L6 AND L5 AND L4
L8      0 S L4 AND L6
L9      0 S L6 AND PREVENT DENATURATION
L10     0 S L6 AND STABILISING MOLECULE

```

=> s p53 and stabiliz molecule

MOLECULE IS NOT A RECOGNIZED COMMAND

The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s p53 and stabil? molecule

```

30076 P53
962138 STABIL?
46847 MOLECULE
145652 MOLECULES
186045 MOLECULE
      (MOLECULE OR MOLECULES)
2198618 MOL
590871 MOLS
2520191 MOL
      (MOL OR MOLS)
2559007 MOLECULE
      (MOLECULE OR MOL)
640 STABIL? MOLECULE
      (STABIL? (W) MOLECULE)
L11     2 P53 AND STABIL? MOLECULE

```

=> d L11 1-2 ibib,abs

L11 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2003:133296 CAPLUS

DOCUMENT NUMBER: 138:166255

TITLE: Stabilization of the native conformation of a mutant
tumor suppressor protein p53 and other
mutant proteins using CDB3 peptide and other
biomolecules and application to treatment of cancer
and other diseases

INVENTOR(S): Friedler, Assaf; Fersht, Alan

PATENT ASSIGNEE(S): Medical Research Council, UK

SOURCE: PCT Int. Appl., 73 pp.

DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

CODEN: PIXXD2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003014144	A2	20030220	WO 2002-GB3668	20020809
WO 2003014144	A3	20031127		
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW			
RW:	GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG			
EP 1414846	A2	20040506	EP 2002-749128	20020809
R:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK			
US 2005008653	A1	20050113	US 2004-775679	20040210
PRIORITY APPLN. INFO.:			GB 2001-19557	A 20010810
			GB 2001-27917	A 20011121
			GB 2002-10740	A 20020510
			WO 2002-GB3668	W 20020809

AB We disclose a method of stabilizing the native state of a polypeptide, the method comprising exposing the polypeptide to a **stabilizing mol.** capable of binding to the polypeptide at a site which at least partially overlaps a functional site in its native state. The authors describe the isolation and identification of a stabilizing peptide CDB3, which is capable of binding the tumor suppressor protein **p53** near its DNA binding site, and stabilizing the native form of the protein. Since the binding of DNA itself stabilizes **p53** core domain, and it binds very tightly, stabilization by a peptide such as CDB3 is needed only for mutants where DNA binding is impaired because mutant **p53** is in denatured conformation. Once the protein has bound DNA, the peptide is not needed any more. The ability of CDB3 to induce refolding of **p53** core domain, together with the observation that DNA can displace it from **p53**, led the authors to propose the a "chaperone" mechanism for rescuing a denatured oncogenic protein: CDB3 binds only the native state of the oncogenic protein which is able to bind DNA, probably immediately on biosynthesis, and therefore shifts the equilibrium towards the native state. Then DNA can bind the protein, displacing the peptide, which is free again to bind another protein mol. Exemplary design of potential **P53** core domain binding peptides, screening of the CDB peptides for binding **p53** core domain, identification of the **P53** core domain binding peptide CDB3, characterization of CDB3-**P53** core domain binding and binding of fluorescein-labeled CDB3 are reported. **Stabilizing mols.** and/or compns. of the invention can be used in the treatment of any animal or human disease where errors in protein conformation, folding and aggregation contribute to the disease. Examples include cancer, cystic fibrosis and neuro-degeneration. In a particularly preferred embodiment, the disease is cancer.

L11 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2000:82983 CAPLUS

DOCUMENT NUMBER: 132:277343

TITLE: Analysis of JNK, Mdm2 and p14ARF contribution to the regulation of mutant **p53** stability

AUTHOR(S): Buschmann, Thomas; Minamoto, Toshinari; Wagle, Nikhil;

Fuchs, Serge Y.; Adler, Victor; Mai, Masyoshi; Ronai, Ze'ev
CORPORATE SOURCE: Ruttenberg Cancer Center, Mount Sinai School of
Medicine, New York, NY, 10029, USA
SOURCE: Journal of Molecular Biology (2000), 295(4), 1009-1021
CODEN: JMOBAK; ISSN: 0022-2836
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Identification of Mdm2 and JNK as proteins that target degradation of wild-type (wt) p53 prompted the examination of their effect on mutant p53, which exhibits a prolonged half-life. Of five mutant p53 forms studied for association with the targeting mols., two no longer bound to Mdm2 and JNK. Three mutant forms, which exhibit high expression levels, showed lower affinity for association with Mdm2 and JNK in concordance with greater affinity to p14ARF, which is among the stabilizing p53 mols. Monitoring mutant p53 stability in vitro confirmed that, whereas certain forms of mutant p53 are no longer affected by either JNK or Mdm2, others are targeted for degradation by JNK/Mdm2, albeit at lower efficiency when compared with wt p53. Expression of wt p53 in tumor cells revealed a short half-life, suggesting that the targeting mols. are functional. Forced expression of mutant p53 in p53 null cells confirmed pattern of association with JNK/Mdm2 and prolonged half-life, as found in the tumor cells. Over-expression of Mdm2 in either tumor (which do express endogenous functional Mdm2) or in p53 null cells decreased the stability of mutant p53 suggesting that, despite its expression, Mdm2/JNK are insufficient (amount/affinity) for targeting mutant p53 degradation Based on both in vitro and in vivo analyses, the prolonged half-life of mutant p53 depends on the nature of the mutation, which either alters association with targeting mols., ratio between p53 and targeting/stabilizing mols., or targeting efficiency. (c) 2000 Academic Press.

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